

A tall order: improving child linear growth

Citation for published version (APA):

Nseluke, M. H. (2018). *A tall order: improving child linear growth: diets, transitions and maternal education*. [Doctoral Thesis, Maastricht University]. Boekenplan. <https://doi.org/10.26481/dis.20180619mn>

Document status and date:

Published: 01/01/2018

DOI:

[10.26481/dis.20180619mn](https://doi.org/10.26481/dis.20180619mn)

Document Version:

Publisher's PDF, also known as Version of record

Please check the document version of this publication:

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Summary

This PhD dissertation titled *A Tall Order: Improving Child Linear Growth: Diet, Transitions and Maternal Education*- studies the association between diet, maternal education and stunting. The study further assesses determinants of dynamics in childhood growth. Stunting is a sign of chronic malnutrition. Stunted children suffer poor health, poor schooling, as adults fail to contribute to or benefit socioeconomically and recycle into poverty. As an indicator, stunting reflects children's well-being and is a reflection of social inequalities (de Onis & Branca, 2016). Subsequently, global health and development goals set targets to reduce stunting by 2030. As stunting persists, affecting 155 million children (UNICEF, 2017), it is acknowledged that strategies that are effective for preventing stunting are still unclear because the extent to which different determinants impact linear growth have yet to be elucidated.

Conceptually, addressing stunting is complex (C. P. Stewart, L. Iannotti, K. G. Dewey, K. F. Michaelsen, & A. W. Onyango, 2013; UNICEF, 1990). On one hand diet, especially dietary diversity of ASF, is considered, a central pillar supporting linear growth (Adelheid W Onyango et al., 2013). In most cases food groups predict nutrient adequacy (Arimond et al., 2010). Yet, dietary inadequacy is such a common occurrence in resource poor settings, prompting suggestions to supplement local diets (Schönfeldt, 2012). On the other hand, maternal education is proposed as the single most factor differentiating behaviors and decisions on child care practices. However, the direct impact of maternal education on linear growth is less studied in developing countries and the impact is attenuated by levels of schooling. Adding to the complexity of research on determinants of child linear growth, is the debate on whether, once stunted, children transition back and forth the stunting cut off. The term "recovery" from stunting is contested as discussed in Chapter 1. However, the use of cut-off points is required to determine the limits of 'normality' (de Onis

& Branca, 2016) and risk to morbidity (Anna Lartey, 2015; Prendergast & Humphrey, 2014).

The principle aim of this dissertation is, therefore, to assess the role of local diets and maternal education on stunting and to study the determinants of childhood growth transitions in Malawi.

In Malawi, stunting is a pressing problem affecting 1.4 million children (UNICEF, 2017). Nearly half of all child deaths and a third of child illnesses in Malawi are associated with stunting (ECA & NEPAD, 2013; Jan Meerman, 2014). The study uses two rich sources of national wide datasets; a Malawi Welfare Monitoring Survey panel dataset (n=530) and two waves of Malawi Demographic and Health Survey cross sectional dataset (women and children n=10,086 and n=14,190 respectively). The research adopts various quantitative analytical techniques: regression models, random effects, probit models and Regression Discontinuity Design and use of instrumental variables. Both data sets used in this analysis are anonymous and exempted from institutional ethical review. The data is publicly available and can be downloaded after registration with the National Statistics office of Malawi from <http://go.worldbank.org/> and ICF at <http://www.measuresdhs.com> (National Statistical Office/Malawi & ICF, 2017).

The four chapters underpinning the aims of the dissertation are summarized as follows;

Chapter 1 introduces the dissertation with the discussion on the scale and consequences of stunting. It then justifies the choice for assessing the role of diet and maternal education on stunting and the determinants of child growth transitions. The chapter presents the empirical debates on diet, maternal education and growth patterns. Some background information about Malawi, the topic of the dissertation, is presented as well. The study relevancy, arising research gaps and research questions are presented and then captured in the four empirical chapters of the dissertation, below.

Chapter 2 systematically reviews literature on impact of ASF on a local non-diverse diet among children aged 6-59 months. The research question which the chapter sought to answer is: what is the evidence on impact of locally available foods, especially ASF, on stunting? The reviewed RCTs investigated a food based supplementation approach using locally available animal source foods and their impact on stunting. Only studies from Sub Saharan Africa were included in order to ensure the review contained similar local food contexts. Studies using mineral and vitamins supplementation were excluded. The search period was from 2000 to January 2017. The review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) and the methodological strength of the studies was evaluated using a commonly proposed criteria (Brannon et al., 2014; Moher & Tricco, 2008). The criteria assess the clarity in study objective, if questions are answerable and protocol, prere-specified eligibility inclusion criteria, the reproducible methodology for stated evidence, critical statement of findings and their validity, and structured presentation of findings.

The RCTs found diverse effects of ASF supplements on child growth, micronutrients and morbidity outcomes. Only milk and eggs supplementation appear to increase linear growth in one study and, in general, other ASF did not show effect. A possible explanation for these results may be due to the fact that in all studies, stunting and morbidity were present by age six months in the study children and worsened over time. Another explanation is that stunting cannot be captured well by attempting to study the effects of a single food item only on stunting. To develop a full picture the dissertation undertook to further investigate on how food groups from local diets in a non randomised setting are associated with linear growth while controlling for underlying factors. In addition, age of child, maternal education and wealth as factors that might influence linear growth were interacted.

Therefore, **Chapter 3** sought to answer a follow up question, which is: what is the association between the quantity and frequency of the consumption of food groups and linear growth? The analysis uses RE regression models to

assess associations with the main outcome since the interacted variables were expected to differ in their effect and maternal education remained constant over time. The main independent variables were food groups and individual foods. In the analysis, foods are grouped from a list of eighty foods found in the dataset. The importance of milk and eggs from RCTs is consistently supported by the findings in chapter 3. Overall, compared to plant based food groups, ASF food group (milk and eggs) emerged as important. This is despite being consumed in small amounts, twice to three times per week. Staples remain dominant in diets. These findings match those found in earlier studies(Theron et al., 2007). The main recommendation for child feeding strategies in Malawi is to promote milk and eggs, and to consider staple fortification since stunting is associated with micronutrient deficiencies, in literature. Chapter 3 further provides evidence on per capita quantity trends and frequency of food groups' consumption. This finding can be basis to benchmark quantities for dietary guidelines in the Malawi context.

One aspect that arose from Chapter 2 is that stunting in the study group worsened and morbidity was common. In Chapter 1 we also summarized the discussion about the (ir)reversibility of stunting. In this regard, **Chapter 4** examines stunting transitions. The premise is that if there is a reduced risk to morbidity when children shift up the stunting cut off, then a question being asked in Chapter 4 is: what are the growth transition probabilities in early childhood, and what are their determinants? Besides the role of diet in Chapter 2 and 3, we sought to explain what determines shifts in the child growth patterns. The analysis used a Markov model to explain current nutrition status, using absolute changes in z scores (WH0, 2006), conditioned on explanatory variables and the child's past state at baseline. We are mindful of the debate on what constitutes recovery as well as the many definitions of catch up growth, which is not being implied in this finding. Rather we demonstrate children that may or maynot be atrisk, if normality is a state above the cut off in literature(de Onis & Branca, 2016).

The findings show that bidirectional growth transitions occurred among children in Malawi. The findings of this research are in line with other studies that reported that children become non-stunted in Malawi and as documented elsewhere. This finding is significant because the analysis uses a panel that followed the same children who lived under the same household conditions. However, while six in ten children transitioned positively, a third of the children remained in the same state from 2010 to 2013, explaining the difficulty of transitioning. Nearly one in five children worsened. This result is nonetheless encouraging because it shows that there is a possibility to minimise the risk to morbidity and possibly to overcome any linear growth deficit, in the long term. The conditions under which positive growth transitions occurred were participation in nutrition programs, increased maternal education, wealth status, absence of illness and a diverse diet. Worsening was predicted by a younger age, being a female child, history of illness and asset poverty. It was beyond the scope of this study to assess the extent of linear growth deficit in the sample and if catch up growth occurred. Hence a future study with more focus on catch up growth on the same cohort of children is recommended.

Chapter 5 is about maternal education causal effect on linear growth. In previous chapters, maternal education was associated with linear growth. But only if the result is causal can we expect maternal education to impact stunting. Therefore, the main question in this chapter is: does higher maternal schooling reduce stunting among their children? We test the hypothesis that increased maternal schooling increases linear growth and reduces stunting. We use the 1994 free education policy reform as a natural experiment. The causal relationship was modelled using the exogenous variability in schooling from the policy reform and a discontinuity in age cut off. In reviewing the literature, the 1994 FPE policy reform favored girls, such as allowing pregnant girls to continue school after giving birth and waving fees for girls at all school levels.

Findings in chapter 5 show that the policy reform yielded the expected results. Illiteracy reduced and mothers exposed to FPE increased their levels of schooling to an average of 5.5 years vs. 4.9 years. Exposed mothers had taller toddlers compared to unexposed mothers. This result suggests that exposure to and increased levels of schooling influence child linear growth outcomes of the next generation. The mechanisms through which increased maternal schooling improves height for age are through dietary intake of milk, child fever treatment, child vaccination and prenatal visits. At maternal and household level, we find that education impacts child growth through increased age at first birth, the reduced likelihood of a small sized baby, reduced maternal agriculture labor participation and the child having an educated father.

Some of the concerning issues to emerge from this chapter is how common teenage marriage, teenage motherhood and dropout rates are, despite a favorable policy. Future research could look into barriers to girl's retention and progression if maternal education is to bring about the expected transformative benefits in their offspring.

Chapter 6 discusses findings which are presented as concluding statements. The overview discussion centers around the four dissertation research questions as follows: 1) What is the evidence on impact of locally available foods, especially ASF on stunting 2) What is the association between the quantity and frequency of the consumption of food groups and linear growth? 3) What are the transition probabilities stunted *versus* non-stunted in early childhood growth, and what are their determinants? 4) Does higher maternal schooling reduce stunting among their children? Below we summarize the concluding statements as follows:

Concluding Statement 1: points to the fact that an adequate and nutritious diet is necessary but not sufficient to promote child linear growth. That determinants of linear growth span across the child, maternal and household levels. In all chapters, the determinants of linear growth pertain to the child's

age, gender and history of illness. At household level, maternal schooling, ASF dietary choices especially milk and eggs, participating in nutrition programs were important, while at household level, wealth status emerged as a factor. This array of factors clearly shows that interventions that focus solely on improving diet are likely to be insufficient. The findings highlight the need for a combined set of interventions that should simultaneously seek to control other underlying determinants which can be mutually reinforcing. The interrelationships found between various factors adds insights to the importance of sector linkages in Malawi.

Concluding Statement 2: states that a transition from a stunted to a nonstunted state is feasible under certain conditions. Our findings suggest that a transition to nonstunted state may enhance individual child movement to the right side of the height distribution for optimal linear growth. This statement follows a view in literature suggesting that risk to illness reduces quickly if the child's HAZ moves above the cut off. In that case, the increase in linear growth reflects the adequacy of the diet and hence also micronutrient status improvement. Improved micronutrient status boosts immunity. Therefore, becoming nonstunted can be expected to minimise the risk of illness in the short term thereby contributing to enhanced linear growth progress and contributing to prevention of occurrence of stunting in long term. This dissertation therefore raises the possibility that health risks for stunted children can be averted by understanding determinants of their growth trajectories motivating a preventive set of interventions.

Concluding Statement 3 says that to be effective, dietary guidelines should promote specific animal source food items from a local diet, that are essential for linear growth. The choice of food items to promote should be based on evidence of what is feasible and commonly consumed. The focus of chapter 2 was to assess the feasibility of supplementing local diets with locally available ASF and their effect on linear growth. In chapter 3, we investigated the effect of various food groups and in what quantities and frequency they were associated with linear growth as a follow up to understand the effects in a non

randomised setting that follow usual dietary patterns. While ASF supplementation was feasible, both chapters show milk and eggs as foods associated with linear growth despite their limited and infrequent consumption of two to three times a week. Both food items appeared to benefit older children from poor households in chapter 3. This result may be explained by the fact that milk is important beyond the breastfeeding age and eggs may be more affordable by poorer households. In any case milk and eggs are foods rich in growth promoting nutrients.

Concluding Statement 4 says that girls' education is an effective strategy for improving child linear growth and reducing stunting in the short and long term. We noticed that maternal education was positively associated with child growth transitions in chapter 3 and feeding patterns in chapter 4. As a follow up to the results above, we tested the causal effect of increased maternal education on stunting. Findings show that the intergenerational consequences of stunting could be minimized by increasing levels of maternal schooling. In the short term, girls with high education delay first births thus preventing adverse effects of teenage pregnancies that contribute to stunting in chapter 5. We show evidence that in the long term mothers with increased levels of schooling have taller children, are less likely to be in the informal labor market, or bear a child with an uneducated father. Supportive behaviors and choices we saw in chapter 5 relate to dietary choices and child illness consultation which contribute to positive linear growth transitions in chapter 3.

To conclude, this dissertation presented an analysis of the relationship between diet, maternal education and stunting. The study also established childhood growth transition patterns and determinants. Collectively, the evidence from the studies suggests that a diverse diet that includes milk and eggs, increasing levels of schooling for girls and participating in nutrition programs may be part of the solutions to prevent stunting.